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Logging in to Dialog

Trying 3106900061...Open

DIALOG INFORMATION SERVICES
PLEASE LOGON:
 ********
ENTER PASSWORD:
□t840lcpq
 ********
Welcome to DIALOG

Dialog leel 00.06.30D

Lat logoff:  28jn00 16:38:09
Logon file001 28jn00 18:43:20
***                                  ***
□dialog

File   1:ERIC  1966-2000/Jun 17
       (c) format only 2000 The Dialog Corporation
*File   1: File has been reloaded. See HELP NEWS 1.

      Set  Items  Description
      ---  -----  -----------
? b 410

>>>'IALOG' not recognized as set or accession number
? set hi ;set hi

       28jun00 18:43:27 User233835 Session D421.1
            $0.40    0.115 DialUnits File1
     $0.40  Estimated cost File1
     $0.05  TYMNET
     $0.45  Estimated cost this search
     $0.45  Estimated total session cost   0.115 DialUnits

File 410:Chronolog(R)  1981-2000 May/Jun
       (c) 2000 The Dialog Corporation plc

      Set  Items  Description
      ---  -----  -----------
?
HILIGHT set on as ''
HILIGHT set on as ''
? b 155, 5, 357

       28jun00 18:44:19 User233835 Session D421.2
            $0.00    0.056 DialUnits File410
     $0.00  Estimated cost File410
     $0.04  TYMNET
     $0.04  Estimated cost this search
     $0.49  Estimated total session cost   0.171 DialUnits

SYSTEM:OS  - DIALOG OneSearch
  File 155:MEDLINE(R)  1966-2000/Aug W3
         (c) format only 2000 Dialog Corporation
*File 155: MEDLINE has been reloaded. Accession numbers have changed.
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  File   5:Biosis Previews(R)  1969-2000/Jun W4
         (c) 2000 BIOSIS
  File 357:Derwent Biotechnology Abs  1982-2000/Jul B1
         (c) 2000 Derwent Publ Ltd

      Set  Items  Description
      ---  -----  -----------
? s modifier(w)locus

           7343  MODIFIER
         126004  LOCUS
      S1    146  MODIFIER(W)LOCUS
? rd

...examined 50 records  (50)
...examined 50 records  (100)
...completed examining records
      S2     90  RD (unique items)
? s s2 and outcrossing

             90  S2
           2039  OUTCROSSING
      S3      6  S2 AND OUTCROSSING
? t s3/6/1-6


 3/6/1     (Item 1 from file: 155)
07026092   92241660
  The effect of linkage and population size on inbreeding depression due to
mutational load.
Feb 1992


 3/6/2     (Item 2 from file: 155)
06813815   92055421
  Coevolution of self-fertilization and inbreeding depression. II.
Symmetric overdominance in viability.
Aug 1991


 3/6/3     (Item 3 from file: 155)
06730578   91301469
  On the evolution of genetic incompatibility systems. VI. A three-locus
modifier model for the origin of gametophytic self-incompatibility.
Jun 1991


 3/6/4     (Item 4 from file: 155)
05432643   89187600
  On the evolution of genetic incompatibility systems. IV. Modification of
response to an existing antigen polymorphism under partial selfing.
Dec 1988


 3/6/5     (Item 1 from file: 5)
07330278   BIOSIS NO.: 000090110180
INBREEDING DEPRESSION WITH HETEROZYGOTE ADVANTAGE AND ITS EFFECT ON
  SELECTION FOR MODIFIERS CHANGING THE OUTCROSSING RATE
1990


 3/6/6     (Item 1 from file: 357)
0251014  DBA Accession No.: 2000-05504
Detection of a genetic locus which can modify a known index phenotype by
   using mutagenized and inbred mouse strains - detection of a
```

phenotype-associated segregating mutation, used to determine the genetic basis of a disease 2000

? t s3/7/6

3/7/6 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

0251014 DBA Accession No.: 2000-05504 PATENT
Detection of a genetic locus which can modify a known index phenotype by using mutagenized and inbred mouse strains - detection of a phenotype-associated segregating mutation, used to determine the genetic basis of a disease
AUTHOR: Dove W F; Shedlovsky A
CORPORATE SOURCE: Madison, WI, USA.
PATENT ASSIGNEE: Wisconsin-Alumni-Res.Found. 2000
PATENT NUMBER: WO 200004186 PATENT DATE: 20000127 WPI ACCESSION NO.: 2000-171274 (2015)
PRIORITY APPLIC. NO.: US 114973 APPLIC. DATE: 19980714
NATIONAL APPLIC. NO.: WO 99US15661 APPLIC. DATE: 19990712
LANGUAGE: English
ABSTRACT: A means of identifying a segregating mutation (SM) by index directed cluster enhanced **modifier locus** and molecule identification method (ICMM), is claimed. This involves outbreeding an inbred founder strain with an index inbred strain, and creating backcross progeny. Progeny exhibiting the outlying phenotype is verified for SM. Also claimed is a similar method involving crossing the founder strain with the index strain, and progeny of that cross carrying the dominant allele are verified for SM. The claims also cover SM identified by **outcrossing** a founder isogenic inbred strain with the index strain, a genetically altered mouse with a genome containing a dominant heterozygous allele conferring an index phenotype. Also covered are a non-human animal selected by these methods, and a means of identifying SM at a genetic locus that modifies the index phenotype in an inbred strain. This can be used to identify a human genetic sequence corresponding to SM at a genetic locus. This is a rapid, focused approach to obtain genes in animal models that influence a medically relevant phenotype to identity the genetic basis of that phenotype. (37pp)

? s s2 and outbreeding

```
            90  S2
           719  OUTBREEDING
     S4      2  S2 AND OUTBREEDING
```

? t s4/6/1-2

4/6/1 (Item 1 from file: 155)
07026092 92241660
The effect of linkage and population size on inbreeding depression due to mutational load.
Feb 1992

4/6/2 (Item 1 from file: 357)
0251014 DBA Accession No.: 2000-05504
Detection of a genetic locus which can modify a known index phenotype by using mutagenized and inbred mouse strains - detection of a phenotype-associated segregating mutation, used to determine the genetic basis of a disease 2000

? t s4/7/1

4/7/1 (Item 1 from file: 155)

DIALOG(R)File 155:MEDLINE(R)

07026092 92241660
The effect of linkage and population size on inbreeding depression due to mutational load.
Charlesworth D; Morgan MT; Charlesworth B
Department of Ecology and Evolution, University of Chicago, IL 60637.
Genetical research (ENGLAND) Feb 1992, 59 (1) p49-61, ISSN 0016-6723
Journal Code: FN2
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Using a stochastic model of a finite population in which there is mutation to partially recessive detrimental alleles at many loci, we study the effects of population size and linkage between the loci on the population mean fitness and inbreeding depression values. Although linkage between the selected loci decreases the amount of inbreeding depression, neither population size nor recombination rate have strong effects on these quantities, unless extremely small values are assumed. We also investigate how partial linkage between the loci that determine fitness affects the invasion of populations by alleles at a **modifier locus** that controls the selfing rate. In most of the cases studied, the direction of selection on modifiers was consistent with that found in our previous deterministic calculations. However, there was some evidence that linkage between the **modifier locus** and the selected loci makes outcrossing less likely to evolve; more losses of alleles promoting outcrossing occurred in runs with linkage than in runs with free recombination. We also studied the fate of neutral alleles introduced into populations carrying detrimental mutations. The times to loss of neutral alleles introduced at low frequency were shorter than those predicted for alleles in the absence of selected loci, taking into account the reduction of the effective population size due to inbreeding. Previous studies have been confined to **outbreeding** populations, and to alleles at frequencies close to one-half, and have found an effect in the opposite direction. It therefore appears that associations between neutral and selected loci may produce effects that differ according to the initial frequencies of the neutral alleles.
? ds

Set	Items	Description
S1	146	MODIFIER(W)LOCUS
S2	90	RD (unique items)
S3	6	S2 AND OUTCROSSING
S4	2	S2 AND OUTBREEDING

? s s1 and mouse

	146	S1
	755861	MOUSE
S5	40	S1 AND MOUSE

? rd

...completed examining records

S6	24	RD (unique items)

? t s6/6/1-24

6/6/1 (Item 1 from file: 155)
10373342 20187915
Predisposition to lung tumorigenesis.
Mar 15 2000

6/6/2 (Item 2 from file: 155)
10326404 20113111

Spatially restricted hypopigmentation associated with an Ednrbs-modifying locus on **mouse** chromosome 10.
Jan 2000

6/6/3 (Item 3 from file: 155)
09870480 99138694
Dystonia associated with mutation of the neuronal sodium channel Scn8a and identification of the **modifier locus** Scnm1 on **mouse** chromosome 3.
Mar 1999

6/6/4 (Item 4 from file: 155)
09669096 98454303
Modifier genes in humans: strategies for identification.
Jan 1998

6/6/5 (Item 5 from file: 155)
09505972 98207250
A high-resolution genetic map of the nervous locus on **mouse** chromosome 8.
Mar 15 1998

6/6/6 (Item 6 from file: 155)
09291309 98011948
Genetic analysis of a quantitative trait in a **mouse** model of polycystic kidney disease.
Oct 1997

6/6/7 (Item 7 from file: 155)
09240462 97434218
Secretory phospholipase Pla2g2a confers resistance to intestinal tumorigenesis [see comments]
Sep 1997

6/6/8 (Item 8 from file: 155)
08864382 97069809
Variants at the secretory phospholipase A2 (PLA2G2A) locus: analysis of associations with familial adenomatous polyposis and sporadic colorectal tumours.
Sep 1996

6/6/9 (Item 9 from file: 155)
08621656 96172827
Modulation of disease severity in cystic fibrosis transmembrane conductance regulator deficient mice by a secondary genetic factor [published erratum appears in Nat Genet 1996 May;13(1):129]
Mar 1996

6/6/10 (Item 10 from file: 155)
08496288 96121384
A curly-tail **modifier locus**, mct1, on **mouse** chromosome 17.
Oct 10 1995

6/6/11 (Item 11 from file: 155)
08421031 96046299

ApcMin: a **mouse** model for intestinal and mammary tumorigenesis.
Jul-Aug 1995

6/6/12 (Item 12 from file: 155)
08313712 95301275
Localization of a murine recessive polycystic kidney disease mutation and modifying loci that affect disease severity.
Mar 1 1995

6/6/13 (Item 13 from file: 155)
07678666 94061981
Genetic identification of Mom-1, a major **modifier locus** affecting Min-induced intestinal neoplasia in the **mouse**.
Nov 19 1993

6/6/14 (Item 14 from file: 155)
06999931 92176249
The Min (multiple intestinal neoplasia) mutation: its effect on gut epithelial cell differentiation and interaction with a modifier system.
Mar 1992

6/6/15 (Item 15 from file: 155)
06568129 91216059
Imprinting by DNA methylation: from transgenes to endogenous gene sequences.
1990

6/6/16 (Item 16 from file: 155)
05498570 89137946
Coevolution of the major histocompatibility complex and the t-complex in the **mouse** . II. Modification of response to sharing of histocompatibility antigens.
Jan 1989

6/6/17 (Item 17 from file: 155)
04232520 83230691
Genetic variability of purine nucleoside phosphorylase activity in the **mouse**: relationship to Np-1 and Np-2.
Apr 1983

6/6/18 (Item 1 from file: 5)
12047141 BIOSIS NO.: 199900327660
Genetic modifiers of polycystic kidney disease in intersubspecific KAT2J mutants.
1999

6/6/19 (Item 2 from file: 5)
11666056 BIOSIS NO.: 199800447787
A cis-acting element that directs the activity of the murine methylation **modifier locus** Ssm1.
1998

6/6/20 (Item 3 from file: 5)
11612745 BIOSIS NO.: 199800394514
The intestinal epithelium and its neoplasms: Genetic, cellular and tissue interactions.

1998

6/6/21 (Item 4 from file: 5)
11300246 BIOSIS NO.: 199800081578
Epilepsy in mice deficient in the 65-kda isoform of glutamic acid decarboxylase.
1997

6/6/22 (Item 5 from file: 5)
10976213 BIOSIS NO.: 199799597358
Localized gene action controlling intestinal neoplasia in mice.
1997

6/6/23 (Item 6 from file: 5)
03724251 BIOSIS NO.: 000024052324
GENETIC VARIABILITY OF PURINE NUCLEOSIDE PHOSPHORYLASE IN THE **MOUSE** RELATIONSHIP TO NP-1 AND NP-2
1982

6/6/24 (Item 1 from file: 357)
0251014 DBA Accession No.: 2000-05504
Detection of a genetic locus which can modify a known index phenotype by using mutagenized and inbred **mouse** strains - detection of a phenotype-associated segregating mutation, used to determine the genetic basis of a disease 2000
? t s6/7/2,4

6/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

10326404 20113111
Spatially restricted hypopigmentation associated with an Ednrbs-modifying locus on **mouse** chromosome 10.
Rhim H; Dunn KJ; Aronzon A; Mac S; Cheng M; Lamoreux ML; Tilghman SM; Pavan WJ
Genetic Disease Research Branch, National Human Genome Research Institute, National Institutes of Health (NIH), Bethesda, Maryland 20892-4472 USA.
Genome research (UNITED STATES) Jan 2000, 10 (1) p17-29, ISSN 1088-9051 Journal Code: CES
Contract/Grant No.: EY10233, EY, NEI
Languages: ENGLISH
Document type: JOURNAL ARTICLE
We have used the varied expressivity of white spotting (hypopigmentation) observed in intrasubspecific crosses of Ednrb(s) mice (Mayer Ednrb(s)/Ednrb(s) and C3HeB/FeJ Ednrb(s)/Ednrb(s)) to analyze the effects of modifier loci on the patterning of hypopigmentation. We have confirmed that an Ednrb(s) **modifier locus** is present on **mouse** Chromosome 10. This locus is now termed k10, using the nomenclature established by Dunn in 1920. The k10(Mayer) allele is a recessive modifier that accounts for almost all of the genetic variance of dorsal hypopigmentation. Using intercross analyses we identified a second allele of this locus or a closely linked gene termed k10(C3H). The k10(C3H) allele is semidominant and is associated with the penetrance and expressivity of a white forelock phenotype similar to that seen in Waardenburg syndrome. Molecular linkage analysis was used to determine that the k10 critical interval was flanked by D10Mit10 and D10Mit162/D10Mit122 and cosegregates with mast cell growth factor (Mgf). Complementation crosses with a Mgf(Sl) allele (a 3-5-cM deletion) confirm the semidominant white forelock feature

of the k10(C3H) allele and the dorsal spotting feature of K10(Mayer) allele. MgF was assessed as a candidate gene for k10(Mayer) and k10(C3H) by sequence and genomic analyses. No molecular differences were observed between the Mayer and C57BL/6J alleles of MgF; however, extensive genomic differences were observed between the C3HeB/FeJ and C57BL/6J alleles. This suggests that alteration of MgF expression in C3H mice may account for the k10(C3H) action on white forelock hypopigmentation. Crosses of Ednrb(s) with Kit(WJ-2) (the receptor for MGF)-deficient mice confirmed the hypothesis that synergistic interaction between the Endothelin and MGF signaling pathways regulates proper neural crest-derived melanocyte development in vivo.

6/7/4 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09669096 98454303
Modifier genes in humans: strategies for identification.
Houlston RS; Tomlinson IP
Institute of Cancer Research, Sutton, Surrey, UK.
European journal of human genetics (ENGLAND) Jan 1998, 6 (1) p80-8, ISSN 1018-4813 Journal Code: B4K
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, TUTORIAL
A number of genetic disorders exhibit inter- and intra-familial variability. Understanding the factors that control the expression of disease genes should provide insight into the fundamental disease processes and will have implications for counselling patients. Different mechanisms can account for this variability, including environmental factors, genotype-phenotype correlations and imprinting. There is also evidence that, in a number of genetic diseases, gene expression is under the control of modifier loci. In cases where the biological basis of the genetic disease is understood, any genes involved in the pathogenic process represent candidate modifier genes which can easily be evaluated. Alternatively, modifiers can be identified through approaches such as **mouse** models. Since modifier genes will generally be common and because of confounding environmental influences, linkage analyses in humans will generally be based upon affected or discordant sib pairs. Discordant sib pairs represent an attractive option for linkage studies, because recurrence rates are high and the reduced survival characteristics associated with severe phenotypes will make the likelihood of obtaining clinical material from two living cases difficult. Furthermore, the use of discordant siblings will select for those siblings which possess sufficient dissimilarity at the **modifier locus** to overcome any shared environmental influence. (42 Refs.)
? ds

Set	Items	Description
S1	146	MODIFIER(W)LOCUS
S2	90	RD (unique items)
S3	6	S2 AND OUTCROSSING
S4	2	S2 AND OUTBREEDING
S5	40	S1 AND MOUSE
S6	24	RD (unique items)

? s s2 and founder

	90	S2
	5219	FOUNDER
S7	1	S2 AND FOUNDER

? t s7/6

7/6/1 (Item 1 from file: 357)

0251014 DBA Accession No.: 2000-05504
Detection of a genetic locus which can modify a known index phenotype by using mutagenized and inbred mouse strains - detection of a phenotype-associated segregating mutation, used to determine the genetic basis of a disease 2000
? s s2 and F(w)1

```
            90  S2
        256031  F
       4751990  1
         17200  F(W)1
S8           0  S2 AND F(W)1
```

? s s2 and progeny

```
            90  S2
         36101  PROGENY
S9           4  S2 AND PROGENY
```

? t s9/6/1-4

9/6/1 (Item 1 from file: 155)
09291309 98011948
Genetic analysis of a quantitative trait in a mouse model of polycystic kidney disease.
Oct 1997

9/6/2 (Item 2 from file: 155)
08621656 96172827
Modulation of disease severity in cystic fibrosis transmembrane conductance regulator deficient mice by a secondary genetic factor [published erratum appears in Nat Genet 1996 May;13(1):129]
Mar 1996

9/6/3 (Item 3 from file: 155)
08313712 95301275
Localization of a murine recessive polycystic kidney disease mutation and modifying loci that affect disease severity.
Mar 1 1995

9/6/4 (Item 1 from file: 357)
0251014 DBA Accession No.: 2000-05504
Detection of a genetic locus which can modify a known index phenotype by using mutagenized and inbred mouse strains - detection of a phenotype-associated segregating mutation, used to determine the genetic basis of a disease 2000
? t s9/7/1-3

9/7/1 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09291309 98011948
Genetic analysis of a quantitative trait in a mouse model of polycystic kidney disease.
Iakoubova OA; Dushkin H; Beier DR
Genetics Division, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts 02115, USA.
American journal of respiratory and critical care medicine (UNITED STATES) Oct 1997, 156 (4 Pt 2) pS72-7, ISSN 1073-449X Journal Code: BZS
Contract/Grant No.: R01DK45639, DK, NIDDK
Languages: ENGLISH

Document type: JOURNAL ARTICLE

The development of a variety of powerful tools for genome analysis has facilitated the ability to genetically map loci which contribute to the variation of a quantitative trait. However, the fact that these traits are often determined as a result of complex genetic interactions has made their analysis considerably more difficult then the molecular characterization of qualitative traits that are monogenic in origin. We have described the use of a novel method of chromosomal exclusion to map the recessive mutation juvenile cystic kidney (jck) to mouse chromosome 11 using an intercross between (C57BL/6J x DBA/2J) F1 jck/+ mice. The severity of polycystic kidney disease (PKD) in the intercross **progeny**, which could be quantitated as a function of kidney size, was significantly more variable than that found in the parental C57BL/6J strain, suggesting that a **modifier locus** or loci introduced from DBA/2J affects expression of jck. Two regions (one from DBA/2J on chromosome 10 and a second from C57BL/6J on chromosome 1) were found to be associated with inheritance of a more severe PKD phenotype. The finding of a highly significant association of inheritance of a C57BL/6J-related locus with disease severity was unexpected since the PKD phenotype in this inbred background is mild. This result suggests that inheritance in the affected F2 mice of loci from the two different parental backgrounds results in the more severe phenotype, presumably as a consequence of a direct or indirect interaction between their protein products. This type of effect, which is an example of genetic epistasis, will make the molecular characterization of loci that contribute to complex traits markedly more difficult than the analysis of monogenic disorders.

9/7/2 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08621656 96172827

Modulation of disease severity in cystic fibrosis transmembrane conductance regulator deficient mice by a secondary genetic factor [published erratum appears in Nat Genet 1996 May;13(1):129]

Rozmahel R; Wilschanski M; Matin A; Plyte S; Oliver M; Auerbach W; Moore A; Forstner J; Durie P; Nadeau J; Bear C; Tsui LC

Department of Molecular Genetics, The University of Toronto, Ontario, Canada.

Nature genetics (UNITED STATES) Mar 1996, 12 (3) p280-7, ISSN 1061-4036 Journal Code: BRO

Languages: ENGLISH

Document type: JOURNAL ARTICLE

Mice that have been made deficient for the cystic fibrosis transmembrane conductance regulator (Cftr) usually die of intestinal obstruction. We have created Cftr-deficient mice and demonstrate prolonged survival among backcross and intercross **progeny** with different inbred strains, suggesting that modulation of disease severity is genetically determined. A genome scan showed that the major **modifier locus** maps near the centromere of mouse chromosome 7. Electrophysiological studies on mice with prolonged survival show that the partial rectification of Cl- and Na+ ion transport abnormalities can be explained in part by up-regulation of a calcium-activated Cl- conductance. Identification of modifier genes in our Cftr(m1HSC)/Cftr(m1HSC) mice should provide important insight into the heterogeneous disease presentation observed among CF patients.

9/7/3 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

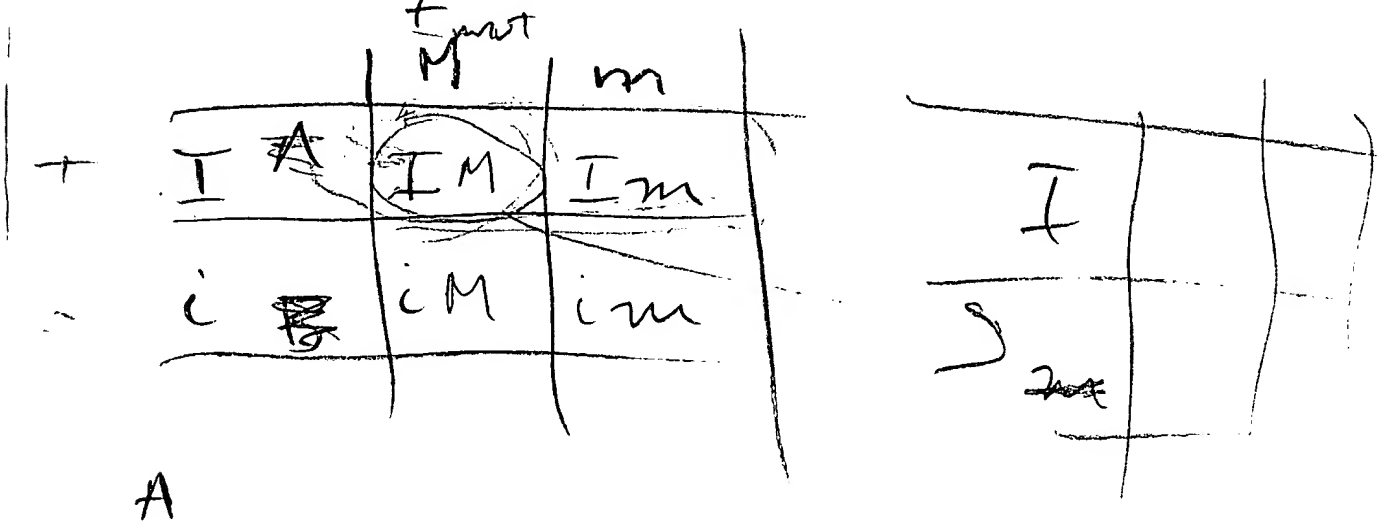
08313712 95301275

Localization of a murine recessive polycystic kidney disease mutation and modifying loci that affect disease severity.

Iakoubova OA; Dushkin H; Beier DR
Genetics Division, Brigham and Women's Hospital, Harvard Medical School, Boston, Massachusetts 02115, USA.
Genomics (UNITED STATES) Mar 1 1995, 26 (1) p107-14, ISSN 0888-7543
Journal Code: GEN
Contract/Grant No.: RO1DK4563902, DK, NIDDK
Languages: ENGLISH
Document type: JOURNAL ARTICLE

We have used a novel method of chromosomal exclusion to map the recessive mutation juvenile cystic kidney (jck) to mouse chromosome 11 using an intercross between (C57BL/6J x DBA/2J) F1jck/ + mice. The severity of polycystic kidney disease (PKD) in the intercross **progeny** was significantly more variable than that found in the parental C57BL/6J strain, suggesting that a **modifier locus** or loci introduced from DBA/2J affects expression of jck. Two regions--one from DBA/2J on chromosome 10 and a second from C57BL/6J on chromosome 1--are associated with inheritance of a more severe PKD phenotype. The finding of a highly significant association of inheritance of a C57BL/6J-related locus with disease severity, with a maximal QTL analysis lod score of 16.8, was unexpected; this result suggests that inheritance of both this locus and at least one DBA/2J locus results in the more severe phenotype, presumably as a consequence of a direct or indirect interaction between their protein products.
? logoff

28jun00 19:01:15 User233835 Session D421.3
$3.15 0.984 DialUnits File155
$0.00 25 Type(s) in Format 6
$1.20 6 Type(s) in Format 7
$1.20 31 Types
$4.35 Estimated cost File155
$4.59 0.820 DialUnits File5
$0.00 7 Type(s) in Format 6
$0.00 7 Types
$4.59 Estimated cost File5
$2.49 0.210 DialUnits File357
$0.00 5 Type(s) in Format 6
$2.20 1 Type(s) in Format 7
$2.20 6 Types
$4.69 Estimated cost File357
OneSearch, 3 files, 2.014 DialUnits FileOS
$0.85 TYMNET
$14.48 Estimated cost this search
$14.97 Estimated total session cost 2.185 DialUnits
Logoff: level 00.06.30 D 19:01:15



QH431. A1 G4

L7 ANSWER 2 OF 4 BIOSIS COPYRIGHT 2000 BIOSIS DUPLICATE 1
AN 1997:42780 BIOSIS
DN PREV199799334768
TI Moml is a semi-**dominant** modifier of intestinal adenoma size and multiplicity in Min/+ mice.
AU Gould, Karen A.; Dietrich, William F.; Borenstein, Natalie; Lander, Eric S.; **Dove, William F. (1)**
CS (1) McArdle Lab. Cancer Res., 1400 University Ave., Madison, WI 53706 USA
SO Genetics, (1996) Vol. 144, No. 4, pp. 1769-1776.
ISSN: 0016-6731.
DT Article
LA English
AB The intestinal tumor multiplicity in mice heterozygous for Apc-Min is strongly modulated by genetic background. On the sensitive C57BL/6J (B6) background, mice develop large numbers of intestinal adenomas. The AKR/J (AKR) strain carries alleles that correlate with a strong reduction in tumor multiplicity. To study the effect of one of these modifiers, Moml, we have generated a mouse line in which the AKR allele of Moml is carried on the sensitive B6 genetic background. This strain was produced by using a marker-assisted selection method to eliminate unlinked AKR alleles more rapidly. The application and efficiency of this method are discussed. We used this strain to determine that Moml affects both tumor multiplicity and tumor size in a semi-**dominant** fashion.

QH573.C38, Editor

Cell 75:631-639, 1993

Mammalian Genome 7(1) 55-58, 1996
7(5) 331-334 1996
9(4) 294-296, 1998
QL738.5. M359

Mol. & Cellular Genetics 240(2) 302-306, 1993

Pestic. Biochemistry & Physiology 53(2) 97-115, 1995

Mol. Teratogenesis Carcinogenesis & Mutagenesis 14(6) 291-302, 1994

Amer. J. Respiratory & Critical Care Med
156(4P1) 572-577